AMERICAN ACADEMY OF PEDIATRICS

Committee on Environmental Health

Environmental Tobacco Smoke: A Hazard to Children (RE9716)

ABSTRACT. Results of epidemiologic studies provide strong evidence that exposure of children to environmental tobacco smoke is associated with increased rates of lower respiratory illness and increased rates of middle ear effusion, asthma, and sudden infant death syndrome. Exposure during childhood may also be associated with development of cancer during adulthood. This statement reviews the health effects of environmental tobacco smoke on children and offers pediatricians a strategy for promoting a smoke-free environment.

EFFECTS OF TOBACCO SMOKE ON CHILDREN

In 1992, 48 million American adults (26.5%) currently smoked cigarettes. A recent national survey indicated that 43% of children 2 months to 11 years of age live in homes with at least one smoker. Because many young children spend a large proportion of their time indoors, they may have significant exposure to environmental tobacco smoke.

Environmental tobacco smoke from cigarettes, cigars, and pipes is composed of more than 3800 different chemical compounds.⁴ Concentrations of respirable suspended particulate matter (particulates of <2.5 μm) can be two to three times higher in homes with smokers than in homes with no smokers.⁵ Cigarette smoking is the most important factor determining the level of suspended particulate matter and respirable sulfates and particles in indoor air.^{6,7}

Passive smoking has a harmful effect on the respiratory health of children.^{4,8,9} This statement reviews the evidence that children exposed to environmental tobacco smoke have higher rates of lower respiratory illness during their first year of life, higher rates of middle ear effusion, and higher rates of sudden infant death syndrome. In addition, children with asthma whose parents smoke have more severe symptoms and more frequent exacerbations.

Passive Smoking and Lower Respiratory Illness

The first effect of passive smoking to be documented in children was an increased rate of illnesses affecting the lower respiratory tract. Cameron¹⁰ reported a positive correlation between the presence of a smoker in the home and the incidence of perceived disease in children.

Harlap and Davies¹ interviewed pregnant women to determine their smoking habits and then studied hospital admissions for infants younger than 1 year. The infants

whose mothers smoked were 38% more likely to be admitted to the hospital for bronchitis and pneumonia than were those whose mothers did not smoke. This increased likelihood was mainly among infants 6 to 9 months of age; admissions increased with the number of cigarettes smoked by the infants' mothers.

Rantakallio¹² showed that, among children younger than 1 year, those with mothers who smoked cigarettes were almost four times as likely to be hospitalized as were the infants of nonsmoking mothers, and the number of hospitalizations increased with the number of cigarettes the mother smoked per day. During the first 5 years of life, pneumonia and bronchitis were about twice as likely and acute nasopharyngitis and sinusitis in the upper respiratory tract were about 1.5 times as likely to develop in children whose mothers smoke.

Colley et al¹³ found a consistent gradient in the incidence of pneumonia and bronchitis in the child's first year of life in relation to the parents' smoking habits. Infants with two parents who smoked were more than twice as likely to have had pneumonia and bronchitis as were infants with parents who did not smoke.

Fergusson et al¹⁴ showed that pneumonia and bronchitis in an infant's first year of life increased with increasing maternal smoking in an approximately linear manner: increases of five cigarettes a day resulted in an increase of 2.5 to 3.5 incidents of lower respiratory illness per 100 children at risk

Passive Smoking and Serious Infectious Illnesses

Berg and colleagues¹⁵ determined that among children 3 to 59 months of age, passive smoking was associated with an almost fourfold risk of a serious infectious illness requiring hospitalization.

Passive Smoking and Middle Ear Effusions

After a case-control study of risk factors for persistent middle ear effusions in Seattle, Kraemer and colleagues¹⁶ reported that children who lived in households where more than three packs of cigarettes were smoked per day were more than four times as likely to be admitted to the hospital for tympanostomy tube placement than were children whose parents did not smoke.

Iversen and colleagues¹⁷ studied children up to 7 years of age in Danish day care centers and demonstrated that middle ear effusion as measured by tympanometry was about 60% more likely to develop in children whose parents smoked. They estimated the overall fraction of middle ear effusion attributable to passive smoking to be 15%.

To determine risk factors for glue ear (serous otitis media), Black¹⁸ performed a case-control study of 150 children 4 to 9 years old undergoing myringotomy in Oxford, England. Children undergoing myringotomy were about 50% more likely to have lived in households where someone smoked than were control children.

Hinton¹⁹ studied 115 children undergoing ear tube insertion for otitis media with effusion and a control group of 36 children from an orthoptic clinic. Children admitted for ear operations were more likely to have at least one parental smoker at home than the children in the control group.

Etzel et al²⁰ studied 132 children in a day care center to determine whether passive smoking was associated with an increased risk of middle ear effusion during the 18-month period between 6 and 24 months of age. In this study, the children were classified as exposed or not exposed to cigarette smoke on the basis of serum cotinine concentrations at 1 year of age. Middle ear effusion was diagnosed with the use of pneumatic otoscopy. The 45 children exposed to environmental tobacco smoke had an average of 7.1 episodes of middle ear effusion between 6 and 24 months of age, whereas the 87 children unexposed to environmental tobacco smoke

had 5.8 episodes during that period. The average duration of middle ear effusion was 28 days among those in the exposed group and 19 days among those in the unexposed group. An estimated 8% of the middle ear effusions were attributed to exposure to environmental tobacco smoke.

Strachan et al²¹ studied the relationship between passive smoking and middle ear effusion in 736 7-year-old school children in Edinburgh. In this study, investigators used objective measures of passive smoking and middle ear effusion, salivary cotinine concentrations, and impedance tympanometry.

Children with type B tympanograms in one or both ears were categorized as having middle ear effusions. The results of this study indicated that detectable salivary cotinine was associated with type B tympanograms, even after adjustment for sex and the type of housing in which the children lived (rented versus owned). The authors estimated that at least one third of the cases of middle ear effusion among children in this age group may have been attributable

to passive smoking.

Owen and colleagues²² monitored 435 healthy children by tympanometry in the home every 2 to 4 weeks until 2 years of age. Of the children, 41% were exposed to household cigarette smoke. The authors found a significant association between the number of cigarettes smoked by household members and the frequency of otitis media with effusion during the second year of life.

Ey and colleagues²³ found that heavy maternal smoking (20 or more cigarettes per day) was a significant risk factor for recurrent otitis media during the first year of life. No association was found with paternal smoking.

Passive Smoking and Asthma

Children with asthma whose parents smoke may have more frequent exacerbations and more severe symptoms. ²⁴⁻³⁵ In one of the few interventions reported in the literature, Murray and Morrison³⁰ demonstrated that if parents expose their children with asthma to less cigarette smoke, the asthmatic symptoms the children have will be less severe.

Passive Smoking and Sudden Infant Death Syndrome

A growing body of evidence links exposure to environmental tobacco smoke to sudden infant death syndrome. ³⁶⁻⁴⁸ This relationship seems to be independent of birth weight and gestational age.

Passive Smoking and Lipid Profiles

Passive smoking has also been reported to alter lipid profiles in adolescents. Feldman et al⁴⁹ studied 391 nonsmoking adolescent students and found that those with elevated plasma cotinine concentrations had an 8.9% greater ratio of total cholesterol to high-density lipoprotein cholesterol and 6.8% lower high-density lipoprotein cholesterol than those with lower plasma cotinine concentrations. This may shed light on the mechanism of increased risk of coronary heart disease in passive smokers.

Passive Smoking and Cancer

Many studies link passive smoking to lung cancer in nonsmoking adults living with spouses who smoke. 50-57 The US Environmental Protection Agency reviewed this subject and concluded that environmental tobacco smoke is a group A human carcinogen, the classification used when sufficient evidence from epidemiologic studies exists to support a causal association between exposure and cancer. A small number of studies have examined the relationship between exposure to environmental tobacco smoke

during childhood and cancer risk. Sandler and colleagues⁵⁸ found that the overall cancer risk was greater for individuals with exposures to environmental tobacco smoke during both childhood and adulthood than for individuals with exposure during only one period. When specific cancer sites or types were considered, Sandler et al⁵⁸ found that leukemia and lymphoma among adults were significantly related to exposure to maternal passive smoke before 10 years of age.⁵⁹

CONCLUSION

Results of epidemiologic studies provide evidence that exposure of children to environmental tobacco smoke is associated with increased rates of lower respiratory illness and increased rates of middle ear effusion, asthma, and sudden infant death syndrome. Exposure during childhood to environmental tobacco smoke may also be associated with development of cancer during adulthood.

RECOMMENDATIONS

- 1. Pediatricians should take smoking histories from parents and guardians of children.
- 2.Pediatricians should inform parents about the health hazards of passive smoking and provide guidance on smoking cessation.
- 3.Pediatricians should set an example by not using tobacco products.
- 4.Pediatricians should promote policies that ensure their offices, waiting rooms, and hospitals are smoke free.
- 5.Pediatricians should urge that sales of all tobacco products be banned in pediatric hospitals and other facilities in which children receive care.
- 6.Pediatricians should work with school boards to ban smoking in schools and on school property, including in teachers' lounges.
- 7.Pediatricians and American Academy of Pediatrics (AAP) chapters should urge their state and local governments to pass legislation prohibiting smoking in child care centers, family child care homes (where care givers care for children who are not their relatives), restaurants, and other public places.
- 8.Pediatricians should work to eliminate cigarette sales from vending machines.
- 9.Pediatricians and AAP chapters should encourage congress and the federal trade commission: (1) to ban all advertising in all media for tobacco products; (2) to sponsor counter advertisements, particularly on television, to inform the public of the dangers of tobacco; (3) to strengthen the health warnings printed on cigarette packages; such messages should specifically warn of the hazards of environmental tobacco smoke; and (4) to increase the federal excise tax on all tobacco products. Higher excise taxes have been shown to deter the purchase of tobacco effectively.
- 10.Pediatricians and AAP chapters should urge congress to dismantle the tobacco price support program.

COMMITTEE ON ENVIRONMENTAL HEALTH, 1996 TO 1997

Ruth A. Etzel, MD, PhD, Chairperson Sophie J. Balk, MD Cynthia F. Bearer, MD, PhD Mark D. Miller, MD Katherine M. Shea, MD, MPH Peter R. Simon, MD

LIAISON REPRESENTATIVES

Henry Falk, MD
Centers For Disease Control And Prevention
Robert W. Miller, MD
National Cancer Institute
Walter Rogan, MD

National Institute of Environmental Health Sciences

CONSULTANT Jim G. Hendrick, MD

REFERENCES

- 1.Centers for Disease Control and Prevention. Cigarette smoking among adults, United States, 1992, and changes in the definition of current cigarette smoking. MMWR. 1994;43:342-346
- 2.Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the US population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988-1991. JAMA. 1996;275:1233-1240
- 3.Schwab M, McDermott A, Spengler JD. Using longitudinal data to understand children's activity patterns in an exposure context: data from the Kanawha County Health Study. Environ Int. 1992;18:173-189
- 4.National Research Council. Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. Washington DC: National Academy Press; 1986:28
- 5. Spengler JD, Dockery DW, Turner WA, et al. Long-term measurements of respirable sulfates and particles inside and outside homes. Atmos Environ. 1981:15:23-30
- 6.Dockery DW, Spengler JD. Indoor-outdoor relationship of respirable sulfates and particles. Atmos Environ. 1981;15:335-343
- 7.Lefcoe NM, Inculet II. Particulates in domestic premises. Arch Environ Health. 1971;22:230-238
- 8.Centers for Disease Control, Center for Health Promotion and Education, Office on Smoking and Health. The Health Consequences of Involuntary Smoking: A Report of the Surgeon General. Rockville, MD: Centers for Disease Control, Center for Health Promotion and Education, Office on Smoking and Health; 1986. US Dept of Health and Human Services publication CDC 87-8398
- 9.Environmental Protection Agency, Office of Research and Development, Office of Air and Radiation. Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders. Washington, DC: Environmental Protection Agency, Office of Research and Development, Office of Air and Radiation;1992. Environmental Protection Agency publication EPA/600/6-90/006F
- 10.Cameron P. The presence of pets and smoking as correlates of perceived disease. J Allergy. 1967;40:12-15
- 11.Harlap S, Davies AM. Infant admissions to the hospital and maternal smoking. Lancet. 1974;1:529-532
- 12.Rantakallio P. Relationship of maternal smoking to morbidity and mortality of the child up to the age of five. Acta Paediatr Scand. 1978;67:621-631
- 13. Colley JR, Holland WW, Corkhill RT. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. Lancet. 1974;2:1031-1034
- 14.Fergusson DM, Horwood LJ, Shannon FT. Parental smoking and respiratory illness in infancy. Arch Dis Child. 1980;55:358-361
- 15.Berg AT, Shapiro ED, Capobianco LA. Group day care and the risk of serious infectious illnesses. Am J Epidemiol. 1991;133:154-163
- 16.Kraemer MJ, Richardson MA, Weiss NS, et al. Risk factors for

- persistent middle-ear effusions: otitis media, catarrh, cigarette smoke exposure, and atopy. JAMA. 1983;249:1022-1025
- 17. Iversen M, Birch L, Lundqvist GR, Elbrond O. Middle ear effusion in children and the indoor environment: an epidemiological study. Arch Environ Health. 1985; 40:74-79
- 18.Black N. The aetiology of glue ear: a case-control study. Int J Pediatr Otorhinolaryngol. 1985;9:121-133
- 19.Hinton AE. Surgery for otitis media with effusion in children and its relationship to parental smoking. J Laryngol Otol. 1989;103:559-561
- 20.Etzel RA, Pattishall EN, Haley NJ, Fletcher RH, Henderson FW. Passive smoking and middle ear effusion among children in day care. Pediatrics.1992;90:228-232
- 21.Strachan DP, Jarvis MJ, Feyerabend C. Passive smoking, salivary cotinine concentrations, and middle ear effusion in 7 year old children. Br Med J.1989;298:1549-1552
- 22.Owen MJ, Baldwin CD, Swank PR, Pannu AK, Johnson DL, Howie VM. Relation of infant feeding practices, cigarette smoke exposure, and group child care to the onset and duration of otitis media with effusion in the first two years of life. J Pediatr. 1993;123:702-711
- 23.Ey JL, Holberg CJ, Aldous MB, Wright AL, Martinez FD, Taussig LM. Passive smoke exposure and otitis media in the first year of life. Pediatrics. 1995:95:670-677
- 24.Burchfiel CM, Higgins MW, Keller JB, Howatt WF, Butler WJ, Higgins IT. Passive smoking in childhood: respiratory conditions and pulmonary function in Tecumseh, Michigan. Am Rev Respir Dis. 1986:133:966-973
- 25.Chilmonczyk BA, Salmun LM, Megathlin KN, et al. Association between exposure to environmental tobacco smoke and exacerbations of asthma in children. N Engl J Med. 1993;328:1665-1669
- 26.Ehrlich R, Kattan M, Godbold J, et al. Childhood asthma and passive smoking: urinary cotinine as a biomarker of exposure. Am Rev Respir Dis. 1992;145:594-599
- 27.Evans D, Levison MJ, Feldman CH, et al. The impact of passive smoking on emergency room visits of urban children with asthma. Am Rev Respir Dis.1987;135:567-572
- 28.Krzyzanowski M, Quackenboss JJ, Lebowitz MD. Chronic respiratory effects of indoor formaldehyde exposure. Environ Res. 1990;52:117-125
- 29.Murray AB, Morrison BJ. Passive smoking by asthmatics: its greater effect on boys than on girls and on older than on younger children. Pediatrics. 1989;84:451-459
- 30.Murray AB, Morrison BJ. The decrease in severity of asthma in children of parents who smoke since the parents have been exposing them to less cigarette smoke. J Allergy Clin Immunol. 1993;91:102-110
- 31.O'Connor GT, Weiss ST, Tager IB, Speizer FE. The effect of passive smoking on pulmonary function and non-specific bronchial responsiveness in a population based sample of children and young adults. Am Rev Respir Dis. 1987;135:800-804
- 32.Oldigs M, Jorres R, Magnussen H. Acute effects of passive smoking on lung function and airway responsiveness in asthmatic children. Pediatr Pulmonol.1991;10:123-131
- 33.Sherman CB, Tosteson TD, Tager IB, Speizer FE, Weiss ST. Early childhood predictors of asthma. Am J Epidemiol. 1990;132:83-95
- 34. Weitzman M, Gortmaker S, Walker DK, Sobol A. Maternal smoking and childhood asthma. Pediatrics. 1990;85:505-511
- 35.Martinez FD, Cline M, Burrows B. Increased incidence of asthma in children of smoking mothers. Pediatrics. 1992;89:21-26
- 36.Bergman AB, Wiesner LA. Relationship of passive cigarette smoking to sudden infant death syndrome. Pediatrics. 1976;58:665-668
- 37.Haglund B, Cnattingius S. Cigarette smoking as a risk factor for sudden infant death syndrome: a population-based study. Am J Public Health. 1990;80:29-32
- 38.Hoffman HJ, Damus K, Hillman L, Krongrad E. Risk factors for SIDS: results of the National Institute of Child Health and Human Development
- Cooperative Epidemiological Study. Ann NY Acad Sci. 1988;533:13-30 39.Lewak N, van den Berg BJ, Beckwith JB. Sudden infant death syndrome risk factors. Clin Pediatr (Phila). 1979;18:404-411
- 40.Malloy MH, Kleinman JC, Land GH, Schramm WF. The association of maternal smoking with age and cause of infant death. Am J Epidemiol. 1988;128:46-55
- 41.Mitchell EA, Scragg R, Stewart AW, et al. Results from the first year of the New Zealand cot death study. NZ Med J. 1991;104:71-76
- 42.Naeye RL, Ladis B, Drage JS. Sudden infant death syndrome: a prospective study. Am J Dis Child. 1976;130:1207-1210
- 43. Steele R, Langworth JT. The relationship of antenatal and postnatal

- factors to sudden unexpected death in infancy. Can Med Assoc J. 1966:94:1165-1171
- 44.Malloy MH, Hoffman HJ, Peterson DR. Sudden infant death syndrome and maternal smoking. Am J Public Health. 1992;82:1380-1382
- 45.Mitchell EA, Ford RP, Stewart AW, et al. Smoking and the sudden infant death syndrome. Pediatrics. 1993;91:893-896
- 46.Schoendorf KC, Kiely JL. Relationship of sudden infant death syndrome to maternal smoking during and after pregnancy. Pediatrics. 1992;90:905-908
- 47.Taylor JA, Sanderson M. A reexamination of the risk factors for the sudden infant death syndrome. J Pediatr. 1995;126:887-891
- 48.Klonoff-Cohen HS, Edelstein SL, Lefkowitz ES, et al. The effect of passive smoking and tobacco exposure through breast milk on sudden infant death syndrome. JAMA. 1995;273:795-798
- 49.Feldman J, Shenker IR, Etzel RA, et al. Passive smoking alters lipid profiles in adolescents. Pediatrics. 1991;88:259-264
- 50.Brownson RC, Alavanja MC, Hock ET, Loy TS. Passive smoking and lung cancer in nonsmoking women. Am J Public Health. 1992;82:1525-1530
- 51.Fontham ET, Correa P, WuWilliams A, et al. Lung cancer in nonsmoking women: a multicenter case-control study. Cancer Epidemiol Biomarkers Prev.1991;1:35-43
- 52.Hirayama T. Cancer mortality in nonsmoking women with smoking husbands based on a large-scale cohort study in Japan. Prev Med. 1984; 13:680-690
- 53.Hole DJ, Gillis CR, Chopra C, Hawthorne VM. Passive smoking and cardiorespiratory health in a general population in the west of Scotland. Br Med J.1989;299:423-427
- 54.Kalandidi A, Katsouyanni K, Voropoulou N, Bastas G, Saracci R, Trichopoulos D. Passive smoking and diet in the etiology of lung cancer among non-smokers. Cancer Causes Control. 1990;1:15-21
- 55.Koo LC, Ho JH, Saw D, Ho CY. Measurements of passive smoking and estimates of lung cancer risk among non-smoking Chinese females. Int J Cancer.1987;39:162-169
- 56.Lee PN, Chamberlain J, Alderson MR. Relationship of passive smoking to risk of lung cancer and other smoking-associated diseases. Br J Cancer. 1986;54:97-105
- 57.Pershagen G, Hrubec Z, Svensson C. Passive smoking and lung cancer in Swedish women. Am J Epidemiol. 1987;125:17-24
- 58.Sandler DP, Wilcox AJ, Everson RB. Cumulative effects of lifetime passive smoking on cancer risk. Lancet. 1985;1:312-315
- 59.Sandler DP, Everson RB, Wilcox AJ, Browder JP. Cancer risk in adulthood from early life exposure to parents' smoking. Am J Public Health. 1985;75:487-492

The recommendations in this statement do not indicate an exclusive course of treatment or serve as a standard of medical care. Variations, taking into account individual circumstances, may be appropriate.

PEDIATRICS (ISSN 0031 4005). Copyright © 1997 by the American Academy of Pediatrics.

No part of this statement may be reproduced in any form or by any means without prior written permission from the American Academy of Pediatrics except for one copy for personal use.